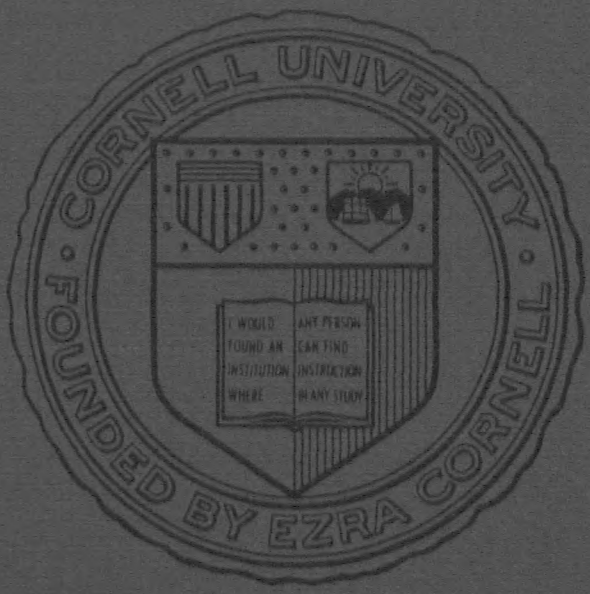
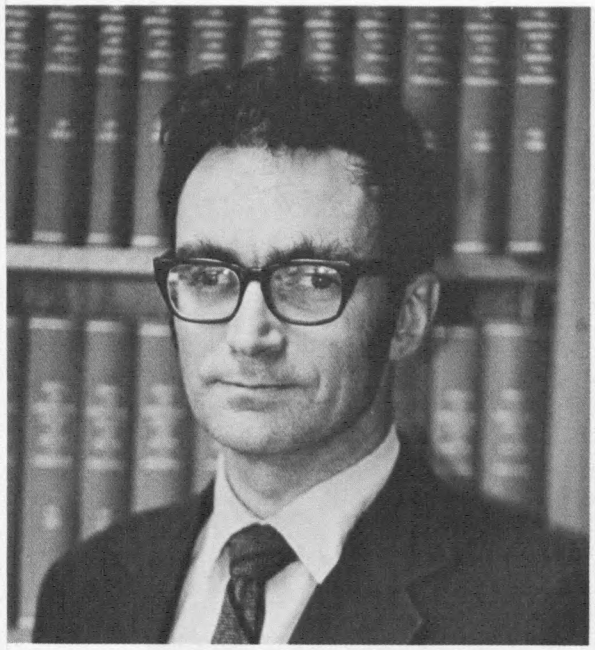
James A. Baker Institute for Animal Health Annual Report 1977



Cornell University, Ithaca, New York

Volume 27

This report honors those
whose generosity sustains the institute's quality and independence,
its high ideals of excellence and responsibility
in the challenge and pursuit of truth.



A Message from the Director

Nineteen seventy-seven has been important for the James A. Baker Institute for Animal Health. This report summarizes some of the significant developments. New construction is under way; new equipment has been added; services and programs are undergoing reorganization; and in the face of escalating costs and shrinking budgets, our faculty and staff have accomplished their mission with dedication and efficiency.

The most exciting developments of this active year may prove to be the advances we achieved in methods for the diagnosis, treatment, and prevention of diseases caused by viruses and other infectious agents. Some of the projects we describe are limited in scope; for example, the attempt to develop a reliable test for the diagnosis of canine brucellosis. Some are broader and deal with a spectrum of questions exploring the effects of nutrition on growth and resistance to infection. But the broader project, too, may have an immediate impact on animal health. A conspicuous example is the ability to modify the expression of hip dysplasia through dietary manipulation early in life. Our studies in search of the mechanism whereby animals develop immunity to parasitic infections further scientific understanding at the most basic level. Other efforts, too, have far-reaching implications, such as the discovery of a mutant strain of canine herpesvirus of diminished virulence.

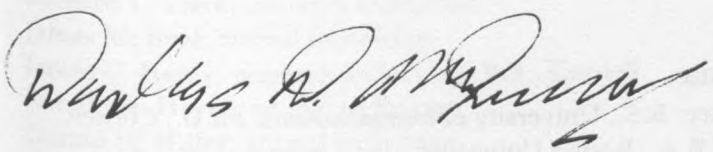
In the span of twenty-seven years the institute has achieved national and international acclaim for its discovery of pathogenic viruses and its leadership in the diagnosis and prevention of diseases caused by a variety of infectious agents. This important work continues as we seek to improve the vaccines currently used by veterinarians to protect dogs against distemper, hepatitis, and other illnesses. But counteracting diseases for which there are no effective vaccines is problematical and complex. Today, advances in the control of infectious diseases can be made only with new information and discoveries gained through the thoughtful application of techniques drawn from the related disciplines of biochemistry, genetics, and cell biology. To remain in the forefront of this research, we have initiated programs that seek a deeper understanding of immune functions. From this we expect to learn more about the fascinating and diverse responses elicited by infectious agents and to manipulate these responses in ways that improve the host's defense.

To ensure the excellence of future research at the institute, we have carefully and critically appraised our resources, our demands for space, and our needs for new equipment. The cost of research mounts annually, threatening our ability to meet even basic requirements. Projections for the years ahead give cause for concern and demonstrate that the institute must secure additional capital and operating funds from both public and private sources. The Surdna Foundation has already responded with a generous gift that is being used to modernize the animal surgery and create two new laboratories and an office. Our future success depends on the ability to attract such outstanding support.

Another major step forward this year was the development of an advisory council composed of distinguished scientists, corporate leaders, and concerned individuals that are deeply interested in animal welfare. It is with great pleasure that we welcome Dr. Richard Johnson, Mr. John Lafore, Mr. Gary Lee, Dr. Irwin Lepow, Dr. Robert Marshak, Mr. John Olin, Dr. Niel Pieper, and Mrs. Richard Scaife as founding members of the council. Their services will be invaluable in appraising our research programs and providing the advice that will ensure a vigorous future for the institute.

Two laboratory reports were published in 1977. Copies of *Canine Brucellosis: Current Status* and *Hip Dysplasia in Dogs: Its Heritable Nature* can be obtained by writing to the institute or by telephoning Mrs. Florence Huth (607/277-3044).

In closing, I wish to thank my colleagues and fellow employees for their commitment to maintaining the institute's tradition of excellence in research, teaching, and public service. I also wish to thank the many individuals whose support, encouragement, and concern have made our achievements and our successes possible.



Douglas D. McGregor, M.D., D.Phil.
Director

Staff

Administration

Douglas D. McGregor, director: B.A., M.D., University of Western Ontario; D.Phil., Oxford University
Neil H. McLain, administrative manager: B.A., Cornell
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Florence C. Huth, secretary
Mary L. O'Brien, account clerk
Ann W. Signore, secretary: Cornell
Douglas S. Robson, consultant in statistics: B.S., M.A., Iowa State University; Ph.D., Cornell

Laboratories

Giralda Laboratories for Canine Infectious Diseases

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Jean C. Joubert, laboratory technician
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Roy Pollock, graduate research assistant: B.A., Williams College

Daynemouth Laboratory for Canine Nutrition

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Chung-sung Chen, graduate research assistant: B.S., D.V.M., National Taiwan University
Alma J. Williams, laboratory technician: B.A., University of Pennsylvania; M.S., Cornell

Biochemistry Laboratory for the Study of Canine Hip Dysplasia

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Peter W. Farrell, laboratory technician: B.S., Cornell

Hadley C. Stephenson Laboratory for Study of Canine Diseases

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Marc H. Langweiler, graduate research assistant: B.S., D.V.M., Cornell
Thomas O. Manning, graduate research assistant: D.V.M., Cornell
James A. Slattery, laboratory technician: B.S., Cornell

Microbiology Laboratory

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Robin G. Bell, research associate: B.Sc., Australian National University; Ph.D., John Curtin School of Medical Research
Thomas W. Jungi, postdoctoral associate: B.A., Kantonsschule Wetzikon, Switzerland; M.S., University of Zurich; D.Phil., University of Basel
Linda C. Dillard, laboratory technician: M.S., Michigan Technological University
Ruth Jungi, laboratory technician
Alan Kay, laboratory technician: A.B., Cornell
Kenneth F. Roberts, laboratory technician: B.S., University of Maine

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Helen A. Greisen, research associate: B.S., M.S., Ph.D., Cornell

Colgate Division for Tissue Culture

Frederick A. Hinman, laboratory technician: B.A., Ithaca College

Glassware Department

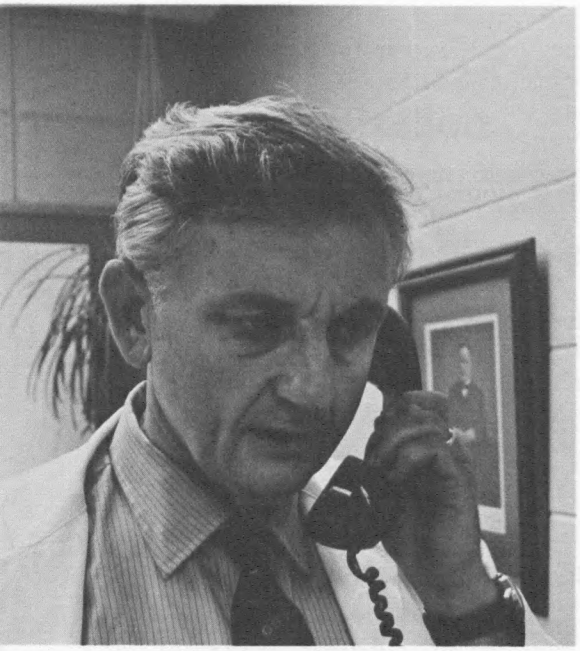
Elizabeth C. Wheeler, supervisor
Judith M. Mead, junior laboratory technician

Animal Care

Charles B. Bailor, animal technician
Roy L. Barriere, animal technician
Michael J. Chapman, vivarium supervisor
Bernard L. Clark, research technician
James M. Ebel, animal technician
James C. Hardy, research technician: B.S., Cornell
Ronald A. Hayes, animal technician
Gerald W. Hiller, animal technician
David L. Watkins, animal technician: A.A.S., State University of New York
James E. Young, animal technician

Maintenance

Edson Wheeler, maintenance supervisor
Merle L. Bower, Jr., custodian
Arthur D. Howser, experimentalist
Eldon Mead, mechanic



Giralda Laboratories for Canine Infectious Diseases

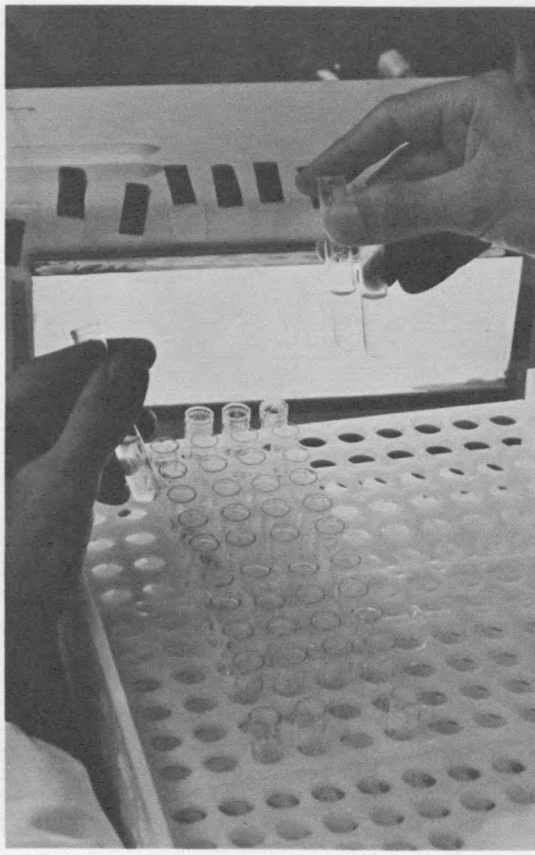
The brucellosis laboratory directed its research toward the causes and diagnosis of canine brucellosis, with important results.

The efforts of laboratory staff to improve current methods of serodiagnosis resulted in significant gains and the development of an important technique: an immunodiffusion (ID) test. The results of laboratory and field evaluations confirm that this new procedure equips us with a more definitive method to determine the presence or absence of canine brucellosis. Using *Brucella canis* cell-wall extract as an antigen, the ID test follows a two-stage procedure on all sera that react in the rapid slide-agglutination test. Dogs that do not respond to standard canine brucellosis testing procedures can be cleared of the stigma of the disease through use of this test and clearly evident reactions of partial identity or nonidentity with the *B. canis* antigen.

Highly prized dogs afflicted with *B. canis* cause personal and economic loss to their owners. The laboratory's success in treating and eradicating this disease in recently infected dogs offers an exciting future for dog owners and the scientific community. Summaries of the current status of methods for diagnosing and treating *B. canis* are available for all who wish to know more about this disease (Baker Institute for Animal Health Publication no. 387).

Until recently specific-pathogen-free (SPF) dogs, such as those raised at the institute, provided the best control for much of our work in canine disease. Investigations have focused on the tissue sites of *B. canis* persistence in chronically infected dogs, the modes of bacterial transmission, the methods of interrupting transmission, and the means by which *B. canis* sustains its growth and survives within mononuclear phagocytic cells. However, with SPF dogs we could not anticipate the problems of heterologous reactions in the field that confound the serodiagnosis of this disease. For example, we recovered at least three bacteria that stimulate the formation of antibodies that cross-react with *B. canis*. To advance our research, we needed naturally infected dogs. We discovered that dogs in Mexico City had a confirmed infection rate of *B. canis* of nearly 12 percent (see Baker Institute for Animal Health Publication no. 386). Using these animals, we can compare the findings of serological procedures with bacteriological results. Through this inquiry we hope to gain insights into the sensitivities and specificities of various laboratory techniques.

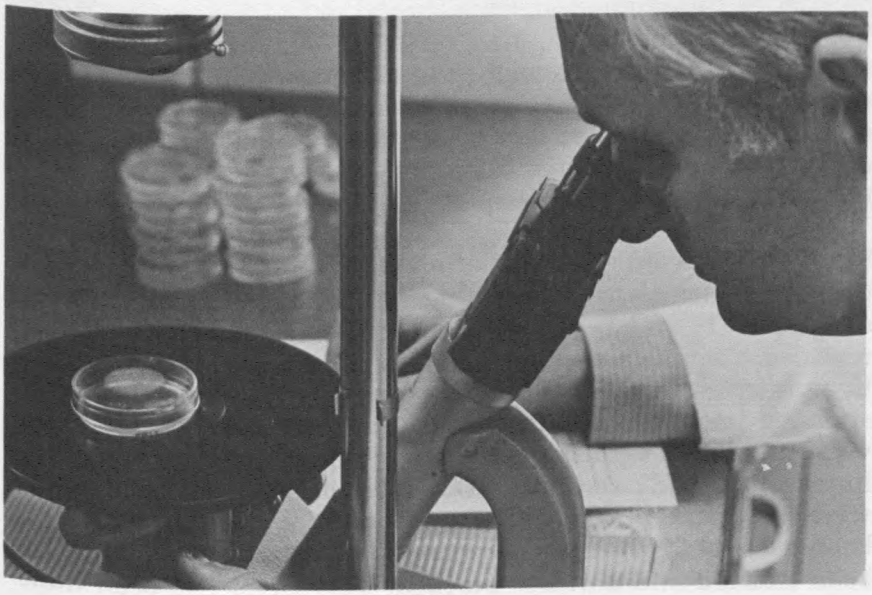
An important offshoot of this research is serological evidence of human infection. Collaborative studies between the institute and the Hospital General de Mexico, S.S.A., are under way to clarify the cause of the extraordinarily high prevalence (13.3 percent) of *B. canis* antibody in sera from Mexico City patients.

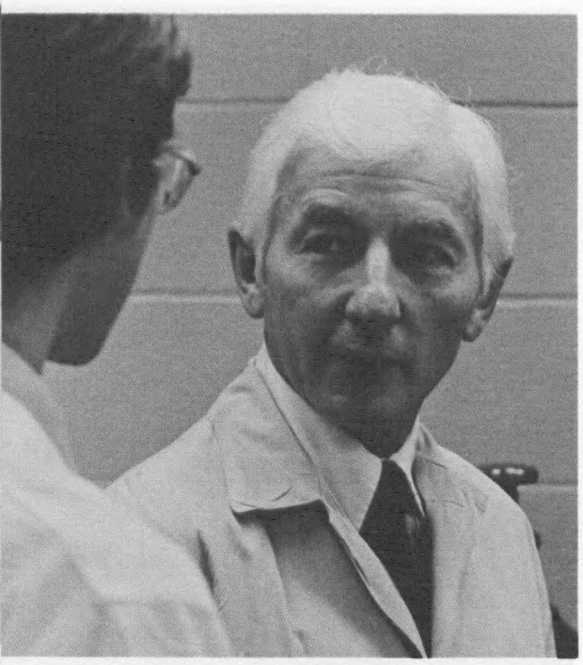


Guided by the important results of past research, the laboratory's interest in herpesvirus continues. A significant step has been the discovery of a suitable strain of canine herpesvirus (CHV) for use as a vaccine. Identified as a small-plaque variant of CHV, the outstanding biological feature of this strain is its virtual lack of pathogenicity for young puppies. Further studies will explore the use of this mutant strain as a vaccine to prevent herpesvirus infection.

We are currently investigating bovine herpesvirus–2 (BHV–2, bovine mammillitis virus), a disease in cattle similar to the cold sore virus in humans (herpes simplex virus–1). The bovine disease becomes clinically manifest under conditions associated with a failure to express cell-mediated immunity (CMI). The most severe eruption of the disease occurs in heifers at the time of parturition or when the weather turns suddenly colder (late summer or autumn). Research demonstrates that the virus can grow at skin temperatures but not internal body temperatures. We are examining the local CMI response to BHV–2 and probing the theory that local changes in skin temperature and increased hormone levels late in pregnancy impede the host's cell-mediated defense against infection.

Leland E. Carmichael





Daynemouth Laboratory for Canine Nutrition

Research efforts in the Daynemouth Laboratory for Canine Nutrition concentrated on the role of vitamin E and related nutrients in the nutrition and well-being of dogs. Vitamin E is known to function primarily as an antioxidant and to contribute toward the maintenance and integrity of cell membranes. To correlate the effects of vitamin E deficiency on measured performance in each biological system, the neuromuscular, hematopoetic, reticuloendothelial, circulatory, and reproductive functions were individually studied.

Our emphasis was on vitamin E deficiency and its influence on immune-response mechanisms. The dog is more resistant than other species to development of muscular dystrophy resulting from a vitamin E deficiency. In our work, no signs of liver necrosis, central nervous disorder, or ocular disease were observed. Diet altered the plasma levels of vitamin E, particularly in rapidly growing puppies, and correlated with erythrocyte fragility, measured by the dialuric acid hemolysis assay. Selenium (Se), sulfur amino acids, and synthetic antioxidants influenced the metabolism of vitamin E, with the net result of delaying or ameliorating expression of clinical vitamin E deficiency.

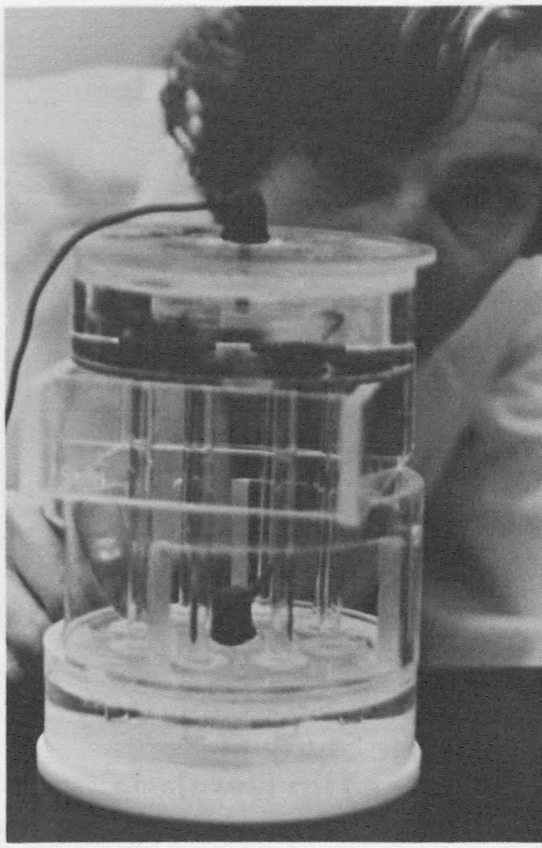
Growth rate and food utilization were significantly affected only when severe deficiencies of both selenium and vitamin E existed. Severely deficient dogs developed a generalized dermatitis, clinically and histologically indistinguishable from that associated with deficiency of essential fatty acid (linoleic acid). However, serum lipid profiles revealed quantities of essential fatty acids to be at or above levels expected for dogs fed a diet containing 4.4 percent corn oil. Thus a clinical dermatitis induced by E-Se deficiency has been recorded. Remission of the clinical signs was not achieved by treatment with prostaglandins; however, the disease did respond to vitamin E therapy as well as to a dietary change from E-free stripped corn oil to E-free stripped lard, with a lowered ratio of unsaturated to saturated fatty acid. These findings suggest that the dermatitis, induced primarily by a polyunsaturated fatty acid toxicity, can be controlled by adequate dietary vitamin E.

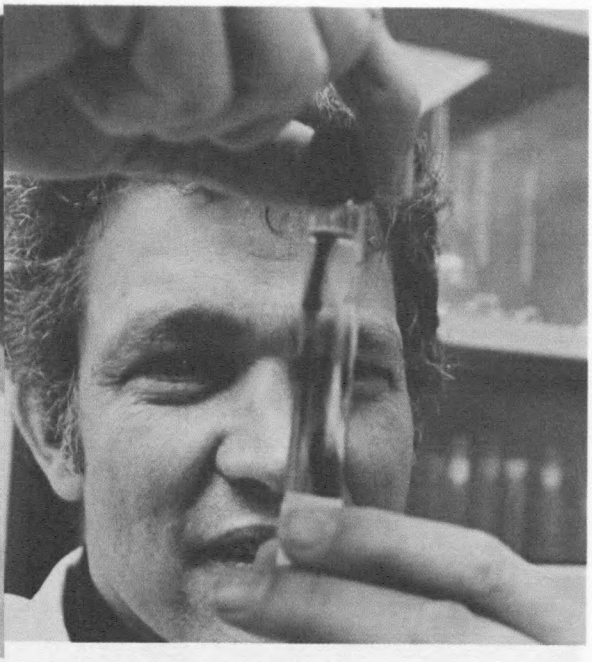
Studies conducted in collaboration with our immunology laboratory clearly demonstrate the influence of vitamin E on the function of specific T- and B-cell response mechanisms. Thus we have revealed a significant new role for vitamin E in canine nutrition. Lymphocytes from E-Se-deficient dogs did not respond, or responded poorly, to in vitro stimulation with mitogens and to in vivo stimulation with sheep red blood cells. Depressing effects on humoral antibody synthesis after vaccination with viral antigens were less marked. The inability of lymphocytes to respond has been associated with the presence of a serum factor, which has been isolated and partially purified. This factor has been trans-

ferred to inhibit the function of lymphocytes in control dogs. Vitamin E, both in vivo and in vitro, transformed or removed the suppressor substance. These studies are being extended to include evaluation of essential fatty acid and prostaglandin roles in the function of immune-response mechanisms, alone and in conjunction with vitamin E.

Other collaborative research described elsewhere in this report is under way to determine the effects of hyperalimentation on organized growth and on the development of degenerative joint disease in dogs.

Ben E. Sheffy





Biochemistry Laboratory for the Study of Canine Hip Dysplasia

Exciting advances leading to a better understanding of the causes of hip dysplasia demonstrate how the research in the biochemistry laboratory can improve the health of dogs and why additional studies are needed.

Canine hip dysplasia is a genetic condition. Nevertheless, manipulating the diet during the first few months of life, when the hip joints are rapidly growing and developing, may have an important impact on the expression of the disease. This possibility is supported by a number of recent observations.

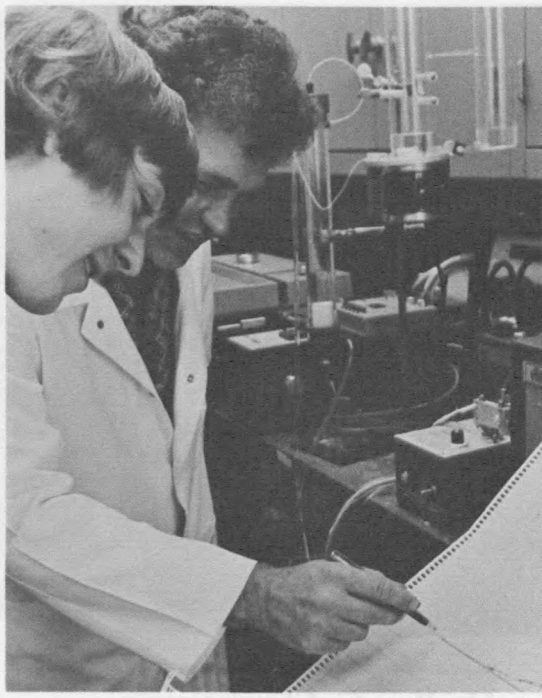
In our experiments puppies with a genetic predisposition for hip dysplasia raised from birth on a low-calorie diet did not develop hip dysplasia, while conventionally reared littermates developed overt disease in six months. In animals whose diet was restricted after weaning the condition was milder and developed later in life. Furthermore, rapid growth in young puppies with normal parents caused hip dysplasia. Other investigators have reported that the rate of weight gain and the ultimate body weight influence the expression of the disease and that overnutrition is associated with a higher-than-expected incidence of hip dysplasia in dogs.

Our studies on the interplay of management conditions and genetic susceptibility lead us to these conclusions:

1. Rapid weight gain during the growth period can bring out hip dysplasia in young dogs, even when their parents have normal hip joints. The offspring of parents with excellent genetic endowment should be less susceptible to the stress imposed by rapid weight gain.
2. Management conditions can reduce or prevent hip dysplasia in puppies whose genetic endowment is in the intermediate range.
3. Dietary restriction alone cannot prevent hip dysplasia in young dogs whose parents are severely afflicted, although it may delay the development of the disease and reduce its severity.

These important findings have two practical applications: (1) Highly prized and valuable dogs can be raised with normal hip joints, even when the genetic predisposition for hip dysplasia exists (with the undesirable result that the genes for hip dysplasia are masked and therefore carried in the breeding stock). (2) Deliberate efforts to promote early rapid growth can reveal disease that might otherwise be masked, so one can select for further breeding only those dogs that maintain the best hip joint conformation.

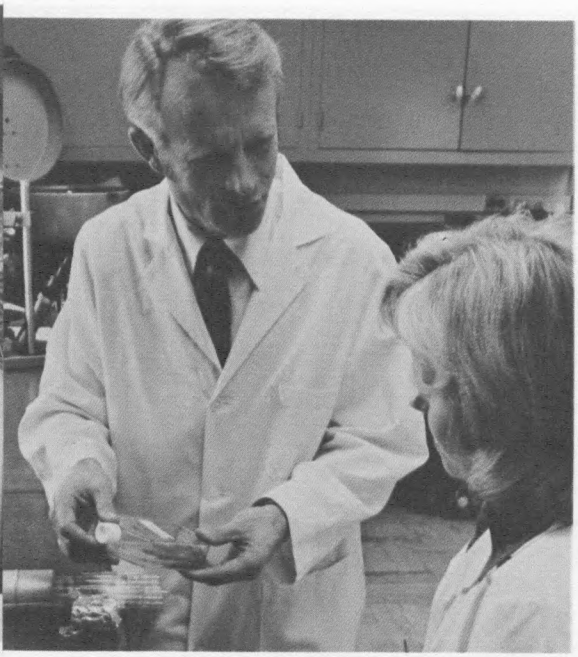
The presence of hip dysplasia often seriously impairs the locomotor function of dogs, largely due to osteoarthritis, an integral part of the dysplastic state. A number of events that either precipitate degenerative hip joint disease in dogs or enhance its severity have been suggested, but the mechanism for its onset and progression must be sought.



Structural and biochemical abnormalities have been observed in the cartilage of osteoarthritic joints. For example, as degeneration progresses, the amount of cartilage decreases in arthritic areas until, in advanced states, collagen and the other constituents of cartilage disappear.

Recently we found increased amounts of procollagen in the osteoarthritic cartilage of young dogs. We propose that procollagen accumulates in this abnormal joint tissue because there is defective conversion of procollagen to collagen. This accumulation may weaken the cartilage and contribute to the erosion of the tissue. By examining the relationship between the accumulation of procollagen in arthritic cartilage and radiologic and pathologic evidence of joint disease, we expect to gain valuable information on the role of metabolic abnormalities in the development and progression of osteoarthritis. Understanding these relationships may lead to ways of preventing the disease.

George W. Lust



Hadley C. Stephenson Laboratory for Study of Canine Diseases

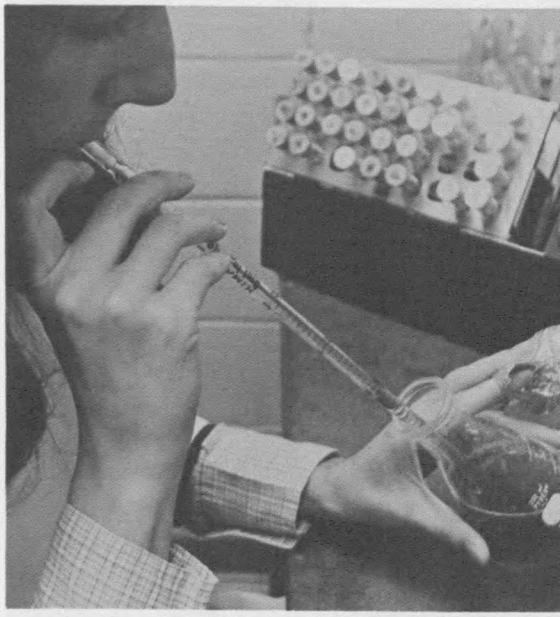
Canine distemper virus (CDV) and other viruses that can produce acute infection occasionally persist in tissues and cause progressive disease, particularly of the central nervous system. Several factors have been implicated to account for viral persistence: the development of temperature-sensitive mutants, the production of defective interfering particles, and the capacity of individual strains of virus to stimulate the formation of interferon. These factors and others have been tested in an effort to explain why individual strains of CDV differ in virulence and in their capacity to cause delayed encephalitis in dogs. Our research revealed a significant difference in the abilities of individual CDV strains to provoke the formation of neutralizing antibody.

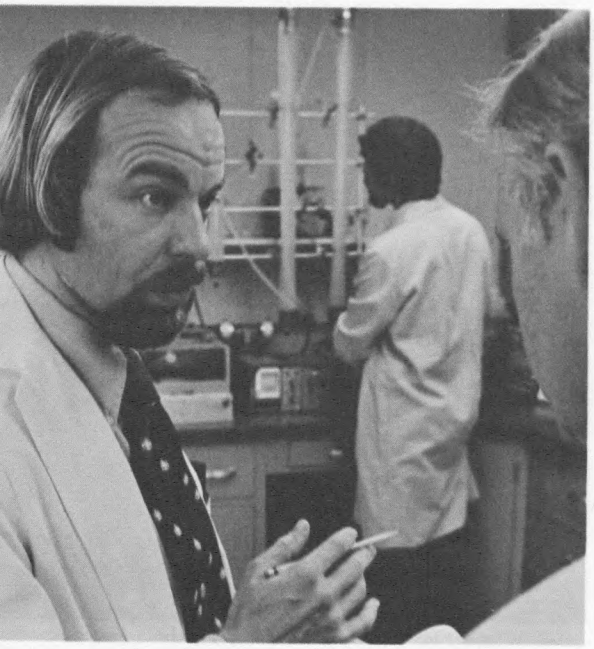
Active CDV serum was tested for its ability to neutralize virulent CDV and attenuated CDV in lung macrophages and Vero cells, respectively. The survival of an infected dog correlated with its capacity to develop neutralizing antibody against both virulent virus and attenuated virus. Dogs fatally infected with the Snyder Hill strain of CDV developed acute encephalitis, and such animals lacked detectable antibody against both virulent and attenuated virus. Dogs with persistent infections caused by an encephalytogenic strain of CDV responded differently. Such animals developed high titers of antibody against virulent virus, but their serum failed to inhibit the growth of attenuated CDV in Vero cells.

These findings suggest that there are antigenic differences between strains of CDV. Evidence to support this was obtained by measuring the serological response to a vaccine strain of CDV. Serum obtained seven days after vaccination neutralized attenuated virus but failed to inhibit the growth of virulent CDV in lung macrophages. Antibody against virulent virus was not detected until ten days after vaccination. These results substantiate the view that neutralizing antibody response to CDV is a critical factor influencing the course of disease. When antibody fails to sterilize tissues, virus spreads to the brain, causing progressive neurological disease.

Other studies sought new insight into the pathogenesis of the brain disease induced by CDV. Systematic analysis of brain lesions brought about by an encephalytogenic strain of CDV provided evidence that the virus is carried into the brain by infected lymphocytes. Sequential experiments using both the light microscope and the electron microscope suggested that CDV spreads from lymphocytes to astrocytes and hence to microglial cells. The latter are often seen next to damaged axons. These findings imply that infected macrophages are involved in the demyelination process. This hypothesis will be tested in vitro using infected macrophages and brain cell explants.

Max J. G. Appel





Oswald R. Jones Laboratory of Immunology

Research continued in the Oswald R. Jones Laboratory of Immunology on the developmental aspects of the cellular and humoral immune response in dogs and cattle and on diseases in which there is an associated immunological deficiency.

The laboratory's efforts to devise practical methods for identifying subclasses of canine lymphocytes and to develop techniques to measure the function of these cells have important implications. This work will further knowledge of the interaction between cells and our understanding of the mechanisms involved in recovery from and protection against infection. We know that defects in the immune capacity can often be attributed to the inability of certain lymphocytes to react. We have shown that the T cell is principally affected in dogs infected with canine distemper virus (CDV). A similar phenomenon is found in humans infected with measles virus. Defects in cell-mediated immunity without an obvious impairment of B lymphocyte function probably occur in other viral diseases.

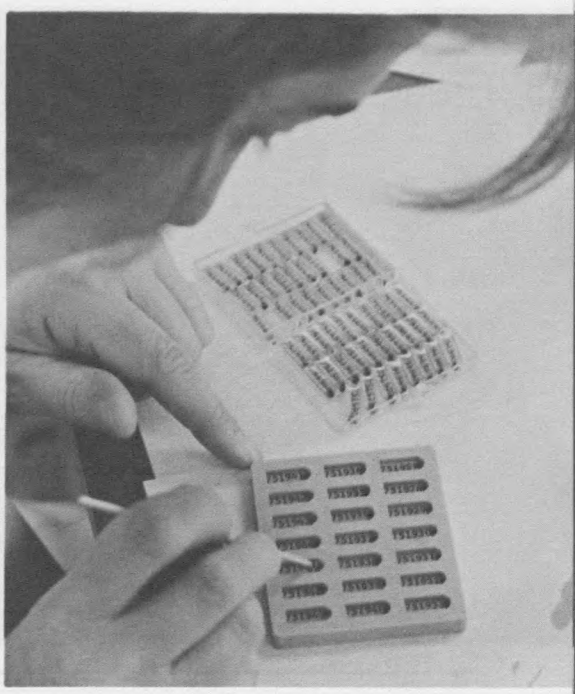
A different line of investigation compared the effect of corticosteroids on the lymphoproliferative response of canine and bovine lymphocytes. These studies were undertaken to reveal the mechanism that reactivates infectious bovine rhinotracheitis (IBR) virus and canine herpesvirus (CHV). Corticosteroids at dosages a hundred to a thousand times those required to inhibit the multiplication of cattle cells had no discernible effect on canine lymphocytes. In spite of the marked resistance of canine lymphocytes to steroid, CHV was reactivated in the dog following a course of steroid treatment.

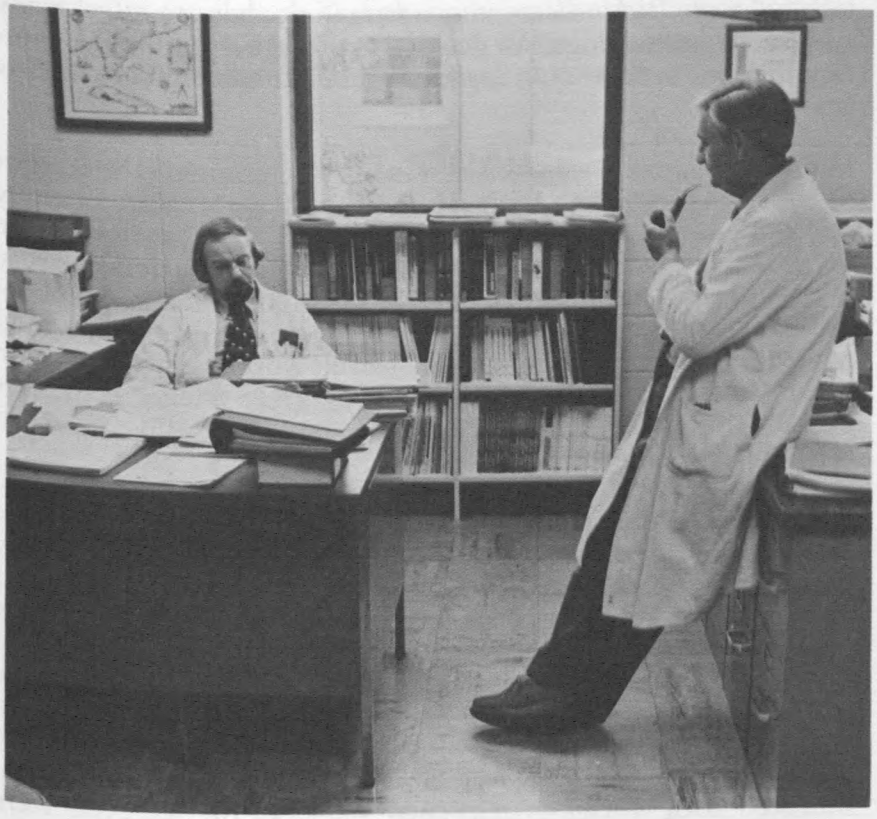
Further exciting work determined the types of lymphocytes present in milk and their possible roles in protection, as well as disease-producing mechanisms, particularly during the critical postnatal period, when the newborn's defenses are weak.

Canine and bovine milk contains cells that are responsive to a variety of T- and B-cell mitogens. The milk of immunized cattle also contains cells that react to tuberculin and IBR virus. Specifically labeled milk cells appeared in a number of tissues, raising the fascinating possibility that antigen-responsive lymphocytes in colostrum and milk enter the general circulation, thereby protecting the newborn against certain infectious agents or potentially causing disease. More studies of lymphocyte migration from the gut into tissues are planned, in an attempt to understand the role lymphocytes have in combating disease. Research is also in progress to determine the role these cells may have in transmitting bovine leukemia virus (BLV) to the newborn and, later, producing virus resulting in infection.

Collaborative research on coonhound paralysis with our colleagues at the New York State College of Veterinary Medicine offers an opportunity to investigate the immunologic and virologic aspects of this disease and compare the results with a similar human disease, the Guillain-Barré syndrome. The human disorder gained nationwide attention when a number of people were stricken after being immunized against swine influenza virus. We are attempting to identify the agent or agents in raccoon saliva that produce paralysis in coonhounds. Not all bitten dogs develop the disease; therefore host factors that influence resistance and prevent occurrence of the disease are being examined.

Ronald D. Schultz





Microbiology Laboratory

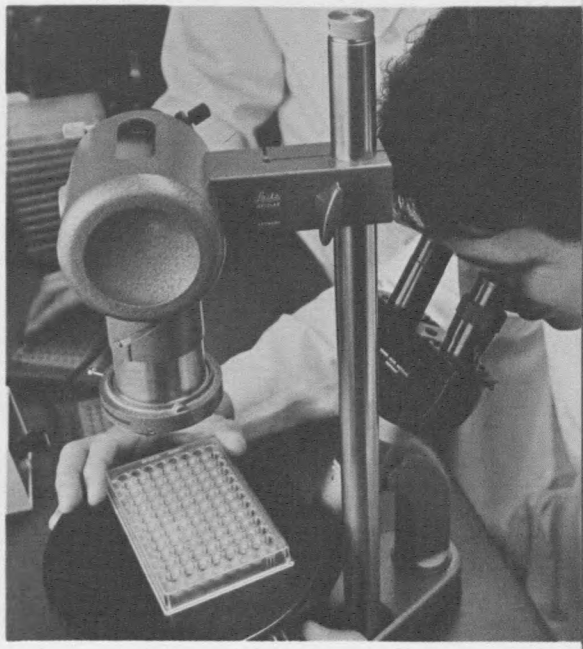
Parasitic infections are among the most common and yet least understood health problems of animals and humans. Parasites inhabit an extraordinary variety of anatomical sites: the intestines and viscera, blood and lymphatic tissue, muscle, and skin. Some of the most important canine infections are those caused by parasites that establish themselves in the intestine. However, before effective ways of preventing disease caused by intestinal parasites can be developed, we must understand how animals become immune to these complex organisms. Information of this kind is obtained in laboratory studies that allow many tests to be designed and carried out.

Our research in the Microbiology Laboratory concentrated on rats infected with *Trichinella spiralis*, a nematode that can infect many species, including dogs. We decided to explore a protective immunity that has been known for fifty years but has not been understood.

Immunity against *T. spiralis* is assessed by determining the number of larvae that establish themselves in the muscle tissue of orally infected animals. Immune subjects harbor fewer larvae than do animals that have not been immunized. This reduction in larvae represents the net effect of several apparently independent responses, some intestinal and others systemic. For example, a rapid rejection response, the expulsion of adult worms, and a reduction in female fecundity all occur in the intestine. Systemic responses include those that are hostile to the tissue-migrating newborn larvae shed by the female.

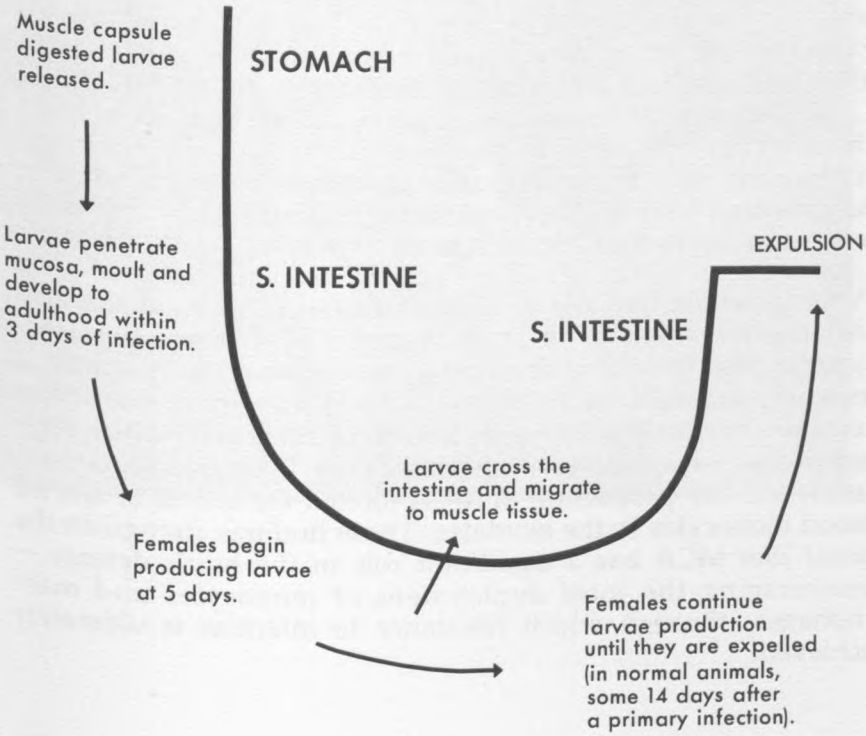
We studied a variety of infections to determine whether different stages of the worm's life cycle are unique either in the antigens they present to the host or in the immune response the host can mount against these antigens. For example, rats infected orally with muscle-stage larvae were given drugs twenty-four hours later that dislodged the parasite from the intestine. Other infections were treated later, when the larvae had matured into adult worms. In still other animals the entire intestinal phase of the infection was avoided by injecting newborn larvae intravenously. Animals immunized with worms in particular stages of the life cycle were challenged with worms in the same or different stages. This cross-infection technique aids in determining whether or not there is variation in the resistance induced by antigens expressed at various stages in the life cycle.

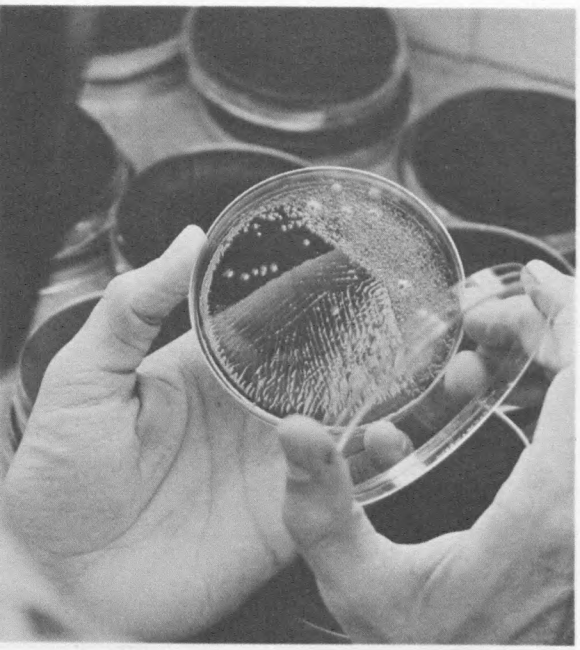
Our efforts, concentrating on intestinal protection, have examined the rapid rejection phenomenon, adult worm expulsion, and antifecundity effects. Direct evidence that adult worm expulsion is mediated by immunological mechanisms is provided by the important observation that this effect can be passively transferred from immune rats to genetically identical nonimmune recipients utilizing either T or B cells from thoracic duct lymph. It has been shown, using the same technique, that the antifecundity effect is also the result of an immunological mechanism. By contrast, attempts to use the classical methodology of serum or cell transfer from immune animals failed to transfer the rapid rejection phenomenon. Protection in this instance is readily recognized by the expulsion from the intestine of 60 to 90 percent of the infecting worms in less than twenty-four hours. This phenomenon is now the subject of an intensive study in which we plan to define the mechanism that achieves rapid antiparasite effects in the intestine.



T. SPIRALIS

STAGES OF DEVELOPMENT IN THE INTESTINAL PHASE





Our research has also concentrated on the underlying mechanism that influences the defensive mechanism whereby mononuclear phagocytes cooperate with lymphocytes in eliminating many infectious agents from the body. Blood monocytes and macrophages exercise their defensive function by phagocytosing and digesting organisms that cannot be disposed of by antibody. They are aided in this process by lymphocytes, in particular T cells, that have a focusing effect on monocytes and enhance the microbicidal capability of macrophages. A critical element in certain bacterial infections is the ability of T cells to recognize the invading organisms and release soluble factors that promote the local accumulation of monocytes.

We and others have shown that a variety of chemical agents can affect the directional movement of monocytes and macrophages, a process called chemotaxis. As chemotactic factors are released in vitro by antigen-stimulated lymphocytes, we suspected that such factors are also generated at sites of bacterial invasion.

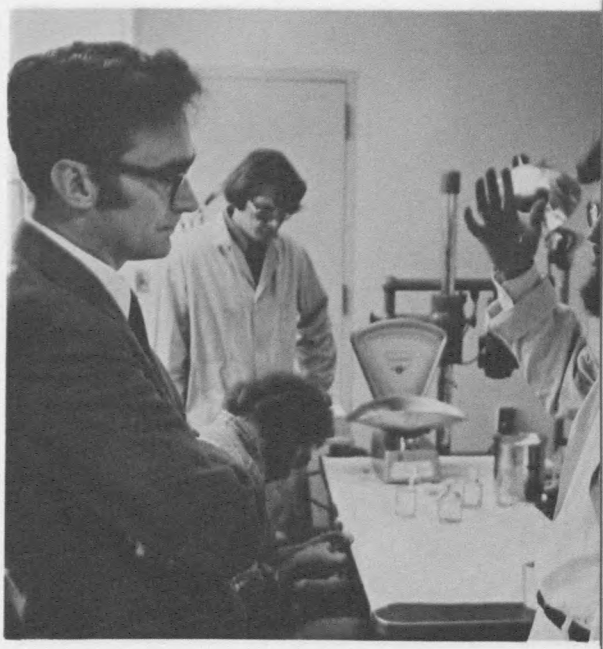
Our experiments used rats immunized against *Listeria monocytogenes*. Intraperitoneal stimulation of infected animals with this organism resulted in the local production of a macrophage chemotactic activity (MCA). Exudate fluids obtained from normal, nonimmunized rats, in contrast to immunized subjects, either were devoid of chemotactic activity or had only feeble activity. Further investigation revealed that the production of MCA is initiated at the site of bacterial invasion. For example, the injection of *L. monocytogenes* at a remote subcutaneous site failed to initiate a chemotactic response in the peritoneal cavity. This observation, in conjunction with the finding that an intraperitoneal injection of bacteria does not alter the chemotactic properties of serum, points once more to the local production of MCA.

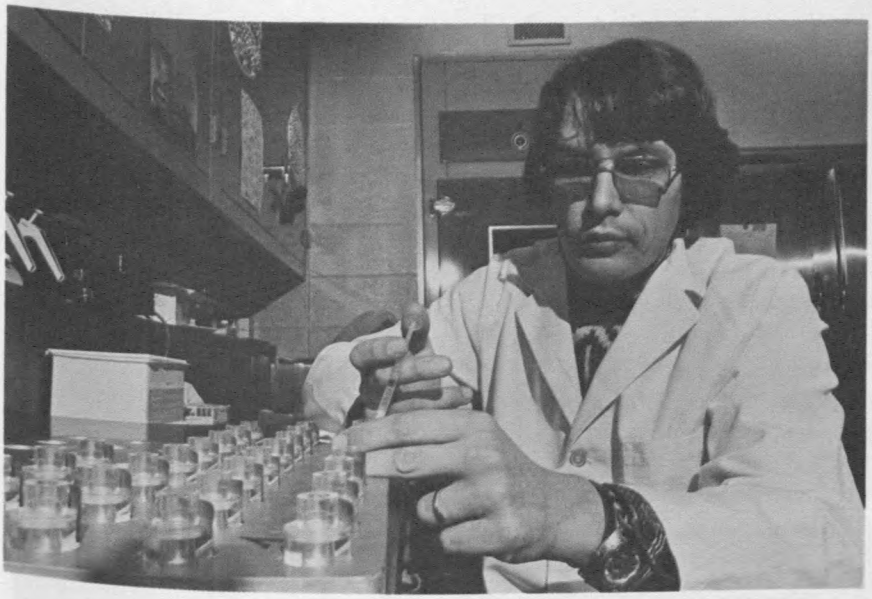
Probing deeper into the underlying mechanism, we discovered that the formation of MCA is triggered by antigen-stimulated lymphocytes. Kinetic studies demonstrated that the appearance, increase, and subsequent decline of MCA in peritoneal exudates coincides or closely overlaps with the local influx of lymphocytes—many of which are recently activated T cells. Lymphocyte accumulation and the production of MCA foretell the arrival of labeled blood monocytes in the exudates. These findings strengthen the belief that MCA has a significant role in the host's defense — encouraging the local deployment of monocytes and macrophages, through which resistance to infection is ultimately achieved.

Additional factors generated by antigen-stimulated T cells attract other lymphocytes into exudates. They play an important part in the host's defense by increasing the intensity of inflammation and thereby attracting monocytes and macrophages into centers of infection.

Our research examined the mechanism whereby monocytes and macrophages perceive chemotactic signals. Using peritoneal exudate cells from germfree rats, we revealed an intrinsic defect in the ability of macrophages to respond to known chemoattractants. This feature may explain why germfree animals fail to express delayed type hypersensitivity and are especially vulnerable to infectious agents that stimulate a cell-mediated immune response. But our findings have other significant implications. Defects similar to those demonstrated in our germfree rats have been observed in newborn subjects, cancer patients, and individuals afflicted with diseases in which there is an associated impairment of cell-mediated immunity. Future experiments will explore these relationships in greater depth.

Robin G. Bell and Thomas W. Jungi





Richard King Mellon Laboratory for Electron Microscopy

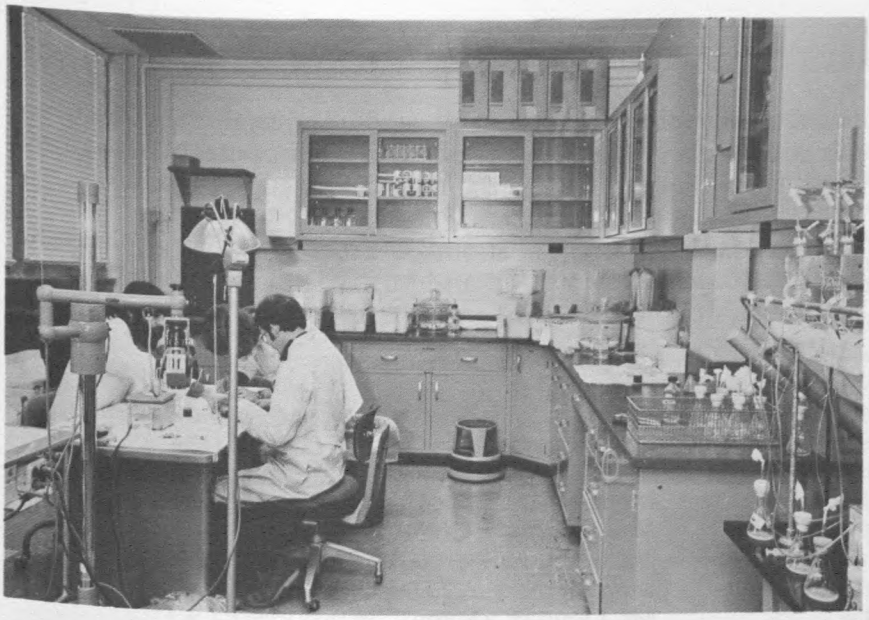
The electron microscopy laboratory participates with other investigators in original research and conducts screening tests for the localization and identification of microorganisms.

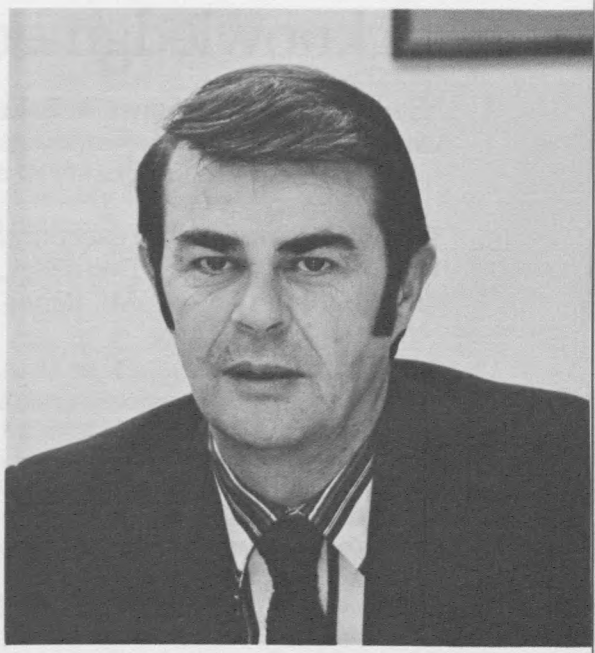
Research on canine distemper encephalitis, a critically dangerous infection, has been furthered by studying brain tissue at arranged intervals. Canine distemper virus (CDV) was identified eight to ten days after the onset of infection in the cytoplasm of lymphocytes surrounding small blood vessels in the meninges, the choroid plexus, the cerebrum, and the brain stem. By twenty days viral nucleocapsids were discovered in astrocytic processes adjacent to endothelial cells in the cerebellum. Little virus was found at this stage, and demyelination was not a conspicuous feature of the disease. By thirty days nucleocapsids showed up in granule cells, macrophages, and glial cells in the cerebellum. Demyelination was widespread. Further studies, aimed at observing the period of twenty to thirty days after infection, are planned. These efforts will determine the sequence of events associated with the spread of CDV in the brain and resultant brain cell injury.

The laboratory assisted in the study of hip dysplasia by describing the ultrastructure of canine articular cartilage and comparing the cartilage of normal and degenerative hip joints. Four distinct layers in the upper 0.5 mm of normal cartilage were found: A layer of fine fibrous material covered the surface. A layer of small (32 nm diameter or less) collagen fibrils were tightly packed in bundles parallel to the surface. There was a layer of less tightly packed collagen fibrils (including fibrils larger than 32 nm). And there was a layer of randomly arranged fibrils more than half of which were 64 nm or larger. The density of fibrils, highest in the surface layer, decreased the deeper the layer in the cartilage.

In moderately advanced lesions of degenerative cartilage a layer of amorphous material appeared on the surface. The tightly packed surface layer of small fibrils was absent. The surface itself was uneven and fissured. At depths comparable to the upper and intermediate layers in normal cartilage the proportion of large fibrils was less. The overall density of fibrils in degenerating cartilage increased with depth into the tissue. Cells flattened parallel to the surface, with relatively large nuclei, were found in the upper layer of normal cartilage. These cells were nonexistent in degenerative cartilage.

The electron microscopy laboratory has become an integral part of the research programs described elsewhere in this report. Morphological studies at both the light- and the electron-microscopic level add important dimensions to the biochemical, immunological, and microbiological methods that are purposefully applied to the study of a variety of canine diseases.





Clockwise from bottom left:
Edward C. Melby, Jr. *(left)*, dean of Cornell's College of Veterinary Medicine, at a recent meeting of the institute's newly formed Advisory Council.

Two members of the Advisory Council, Mr. Gary Lee *(center)* and Dr. Robert Marshak *(right)*, talking with Dr. George W. Lust.

A renovated laboratory, made possible through a gift from the Surdna Foundation.

Neil H. McLain directs the institute's day-to-day functions.

Florence C. Huth's responsibilities include managing the institute's publications.

Acknowledgments

Your interest in the James A. Baker Institute for Animal Health, expressed by your gift, enables us to carry out our day-to-day mission. With your support we can respond swiftly to opportunities as they arise and improve the quality of animal health. Your gift earns the institute's deepest thanks.

In appreciation for their exceptional interest in the institute, we should like to express our gratitude to Mr. and Mrs. Gaylord Donnelley, Mrs. Priscilla M. Endicott, Mr. and Mrs. E. Roland Harriman, Mr. and Mrs. John Lafore, Mr. James A. Moffett, Mr. John M. Olin, and Mr. Robert W. Woodruff.

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Long Island Veterinary Medical Association
(in memory of Dr. Theodore Allen)
Monroe County Veterinary Medical Association
(in memory of Mr. Bruce E. Smallridge)
Veterinary Medical Association of New York City, Inc. (in memory of Dr. Charles E. Fanslau and Dr. Edwin R. Blamey)
Westchester-Rockland Veterinary Medical Association
Western New York Veterinary Medical Association, Inc.
Women's Auxiliary to the Long Island Veterinary Medical Association (in memory of Brian DuBato)
Women's Auxiliary to the New York State Veterinary Medical Society
Women's Auxiliary to the Veterinary Medical Association of New York City, Inc. (in honor of Martha Greenhouse Sasmor)

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Erie Kennel Club
(in honor of Dr. Richard Kester)
Finger Lakes Kennel Club*
First Dog Training Club of Northern New Jersey
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(in honor of Dr. R. A. Faulkner)
Glens Falls Kennel Club, Inc.*
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In Memoriam

Dr. Theodore Allen
Miss Betty Barr
Mr. Thomas E. Berry
Dr. Edwin R. Blamey
Mr. Clarence G. Bradt
Dr. Victor Buencamino
Dr. Charles E. Fanslau
Mr. Charles Georgoulis
Mrs. Catherine Greenwood
Mr. C. Edward Murray, Jr.
Mrs. Helen Ribble
Mr. Bruce E. Smallridge
Dr. Harry M. Zweig

Publications

Publications for the first ten years are listed in the institute report for 1960. Those for each year thereafter appear in the annual report for that year. Since 1960, articles have been numbered consecutively. Some of the following publications have been listed in a previous year's report as *accepted* or *in press*. They are repeated this year, with their original numbers, to record full bibliographic details. Articles completed during the past year are those numbered 369 to 430.

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Ways of Giving

In establishing the institute, of which the Cornell Research Laboratory for Diseases of Dogs is an important part, the Cornell University Board of Trustees authorized the Treasurer's Office of Cornell University to act as custodian of all funds given in support of the institute. As a donor, you are thus assured your gift will achieve the maximum benefit.

There are many ways you can give to advance the work of the institute. Some of these opportunities offer substantial income tax and estate tax benefits.

Checks. All checks should be made payable to Cornell University and mailed to:

Office of the Director
James A. Baker Institute for Animal Health
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for the uses and purposes of the Cornell Research Laboratory for Diseases of Dogs.

Appreciated stocks. Selling appreciated stocks is almost certain to increase your taxes. You gain maximum tax benefits by giving the stocks to Cornell outright and deducting their *full* current market value as a charitable contribution, thus avoiding capital gains tax. The transaction can be completed with maximum speed and at lowest cost by following these steps:

1. Decide what securities you want to give and take the certificate to your bank or broker.
2. Inform your bank or broker you want to make a gift of these shares or securities to Cornell University for the institute.
3. Instruct your bank or broker to telephone the Office of University Investments, at 607/277-0022.
4. Write a note to the Director, James A. Baker Institute for Animal Health, Cornell University, Ithaca, New York 14853, including the name of your bank or broker and the form and size of your gift.

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Deferred giving — income-producing trusts. An income-producing trust enables you to make a meaningful gift to the institute, gain spendable income for life, and derive important tax benefits. A beneficiary may be named to receive this income, too. The institute can offer three plans: the Pooled Life Income Fund for gifts of $5,000 or more, the Annuity Trust, and the Unitrust for gifts of $50,000 or more. Currently, each plan supports an income of about 7 percent a year.

Financial planning involving deferred gifts is a highly complex subject requiring expert advice from your attorney and other specialists. If you are interested in this way of supporting the James A. Baker Institute for Animal Health, please notify the director, who will make arrangements for you to receive more specific information.

Financial Situation

To assure donors that their funds will sustain and advance research on dogs now and in the future, the Cornell University Board of Trustees made a provision for disposal of excess income as follows: "The Institute's income is in excess of its operating expense, and the balance of the funds is added to the Institute's Endowment."

July 1, 1976, to June 30, 1977

Funds Available

Gifts and earnings budgeted	$294,651
State of New York general support	116,625
State of New York dog license fees	144,000
	$555,276

Expenditures

Salaries	$468,384
Operational costs	124,494
	$592,878

Reserves used to balance budget	$ 37,602

Research that involves species other than dogs is supported by other sources.

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